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A NOTE ON A CASE OF TROPICAL TYPHUS OCCURRING IN MADRAS

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Every practitioner in the Tropical and Sub-tropical climate has been faced with the problem of continued fever. Having eliminated the usual causes for this type of fever, there remain a certain proportion of cases in which the diagnosis remains in doubt.

I have recently met with a case of this kind in Madras which was only diagnosed some time after the patient had recovered, and the diagnosis of which explained two other cases that I had seen previously in this Presidency, and which had remained obscure.

CASE—Mr. M.—a European, aged 33, called me in to see him in the middle of December 1934; he complained of fever which had started 4 days before my visit and seemed of an abrupt onset, but without a definite rigor. He, however, admitted feeling "out of sorts" for a few days before this time. Apart from the fever, he complained of headache and pain in the limbs: his past history and family history revealed nothing of interest.

On examination, the patient seemed to be a well-built man, somewhat pale, no jaundice, mucous membranes of good colour, temperature 101° F, pulse 84, respirations 24; the tongue was furred and the spleen palpable on respiration; the abdomen was flaccid without a trace of tenderness, quite unlike the early typhoid abdomen. The urine contained a trace of albumin, but no abnormal deposit. The other systems were normal. A blood slide was taken and *Atebrin* tablets, one, three times a day, prescribed. This was continued for 2 days, but his condition failed to improve and the blood slide was negative for Malarial Parasites, so they were omitted.

Patient was admitted to Hospital on 19-12-34 on the sixth day of his illness. On admission his temperature was 99° F, pulse rate 96, respiration 24. It was found that the thermometer was faulty and his temperature was almost certainly over 101° F. The tongue was rather more furred, the spleen more palpable and the urine still contained a trace of protein. The temperature continued to fluctuate between 101° and 103° and the pulse between 80 and 100 for 6 days after admission and 12 days after the commencement of the fever. The temperature and pulse then gradually settled to normal which has finally reached on the 22nd day, though from the 15th day only minimum rises to 99° in the evening were recorded.

THE RASH: A maculo-papular rash appeared on the abdomen and trunk on the 8th day of the fever, spreading to the arms and hands and to a less extent the face and the legs. The rash was profuse, not petechial; there was no irritation, and the rash had faded by the 15th day after the disease.

THE HEADACHE: This was very severe, but was controlled by 10 to 15 grs. of Aspirin in the day. The headache was distributed over the head, and was associated with marked hyperaesthesia of the overlying scalp. On the 10th day of the fever, the patient complained of an irritable cough, and by the 12th day some signs of congestion were found over the base of the right lung. At this time, the heart was dilated, the apex beat having moved out to beyond the nipple line in the 5th space. The first sound at the apex was soft, but the blood pressure was maintainted at 120/80 mm. Hg. throughout the disease. The patient was definitely toxic between the 9th and 13th day of his illness; after the 14th day of the fever the patient's condition showed a steady improvement, but the headaches did not leave him completely till the 20th day of the disease.

During the fever the bowels had to be kept open by the use of Glycerine enemata, though they began to work naturally when the temperature fell to normal. The lung signs and the cough cleared up by the 15th day; the apex beat returned to its normal situation, the urine cleared, but the spleen remained palpable.

Investigations

Blood slides: Examined on first, seventh and eighth days of the fever; no Malarial parasites found—a slight leucocytosis noted on each occasion and a definite increase of polymorphonuclear cells, most of which showed some immaturity, i. e., the older type of meta-myelocyte.

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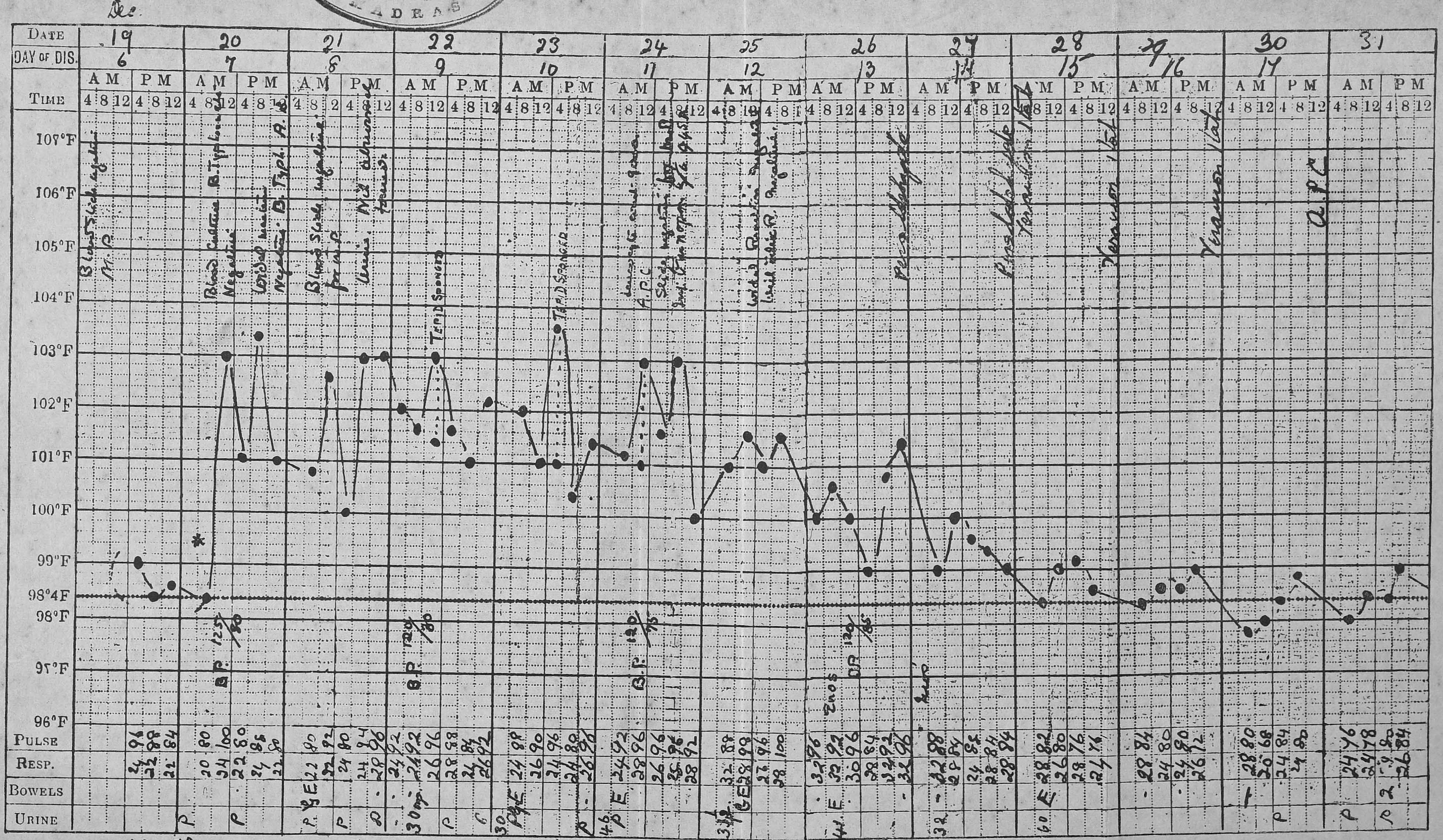

* The thermometer was found to be faulty.

Hoe & Co., Madras.

Blood count:

Total white count 9,000 per c. mm.

Differential count:

Polymorphenuclears	80 per cent
Lymphocytes	12 „
Large Mononuclears	4·5 „
Eosinphiles	1 „
Transitional cells	2 „
Mast cells	0·5 „

Blood culture on the 7th day of the disease *negative* for *B. typhosus* and other organisms. **Widal reaction** *negative* on the 7th day and 10th day. **Weil-Felix reaction** *negative* on the 12th day.

Urine:

Acid,
Sp. Gr. 1012.
Albumin—trace
Deposits—Amorphous phosphates
No casts, pus, or erythrocytes.

Stools showed no abnormality.

The case was seen in consultation with Major McRobert, I.M.S. on the 12th day, and it was thought that, in spite of the negative Weil-Felix reaction, the case most probably belonged to one of the tropical typhus fevers. At his suggestion serum was taken and despatched to the Institute of Medical Research, Kuala Lumpur, F. M. S., to be put up against their standard strains of *Proteus X. 19.* The reply was received at the end of January 1935, that the serum had agglutinated as follows—

Proteus X 19 in 'K' (Kingsbury strain)—1 in 25;

Proteus X 19 'W' (Warsaw Strain)—$\frac{1}{4,400}$

a figure which is diagnostic of a type of typhus known as the *Urban* type.

I examined the patient again 4 weeks after his discharge from Hospital and found him normal in every respect. The spleen could no longer be palpated, no protein found in the urine. It will be seen from the analysis of this case that we have a *continued remittent fever* of roughly 15 days duration which ended by *lysis.* The fever was accompanied by relative bradycardia.

The next most distinctive feature of the case was the rash which was

first noticed on the 8th day of the trouble. It began very profuse and consisted of a discrete maculo-papular eruption. It was not so marked on the lower limbs as elsewhere and had completely disappeared by the 15th day. The spleen was definitely palpable on the 4th day of the onset of the fever. Certain features such as a relative bradycardia, the severe headache, palpable spleen and rash might at first sight suggest enteric fever; the abdomen, however, was quite unlike that of typhoid; the rash was too profuse and the individual macules larger than the rose spots of typhoid fever. The blood count and the negative serological reactions definitely excluded this disease. The possibility of an *Infective Endocarditis* was entertained, but the absence of the petechiae land embolic phenomena, and the course of the fever soon excluded this.

Since seeing this case I could call to mind two other cases of fevers of most probably the same type that have come under my observation in Madras. The first occurred in a European boy of 12 years who had high fever for 5 days when I saw him first; no cause could be found for the fever and the possibility of mild enteric was considered and the patient admitted to Hospital; two days after admission he came out with a profuse eruption that was maculo-papular, and, in his case, was general in distribution, including the legs. After 4 days the rash faded and the patient made an uneventful recovery. At no time was he really ill and he never became toxic. The spleen was just palpable throughout the disease. The fever had settled by the 14th day and the patient has remained in excellent health ever since. In this case there was also a slight leucocytosis and the tests for enteric were negative.

The third case that I saw which in retrospect was, I am almost certain, a case of typhus, occurred in an Indian gentleman of good position, aged 50, who came from the neighbourhood of Trichinopoly. He had fever for some 7 days prior to admission, and on the 13th day of the fever, a profuse papulo-macular rash came out which lasted about 4 days. He was definitely toxic, the spleen was certainly palpable. He had tachycardia throughout his illness and his lungs were full of bronchitic sounds; the teeth were very septic, and his general physique was rather below par. After the 20th day the fever settled by *lysis* and the patient made a complete recovery.

In both the above cases, the Weil-Felix reaction was done; in the first case it was returned negative, and in the second positive, 1 in 200, which was not considered diagnostic.

A NOTE ON TROPICAL TYPHUS FEVER

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Of recent years it has become increasingly evident that diseases of the typhus group are prevalent throughout the Tropics.

Cases of non-epidemic typhus have been reported from North and Central India, from Malaya and Burma, and it is probably only a matter of time before the existence of the disease all over India is proved.

The case recorded in the preceding pages by my colleague Captain R. D. Alexander, I.M.S. is, I believe, the first to be reported from the area in which this JOURNAL circulates.

Another similar case was recently diagnosed on clinical grounds in my wards in the Madras General Hospital.

Mrs. A. P.—a fair Anglo-Indian woman, aged 22 years, was admitted to the Madras General Hospital on 2-1-33 with a history of continuous fever of four days duration. The illness had commenced with shivering, and there was severe headache.

The spleen was just palpable. The abdomen was not tender and was not distended. Repeated blood films were negative for malaria parasites and blood culture taken on admission was negative to enteric group. Throughout the illness the Widal reaction was negative as were also stool and urine cultures.

Towards the end of the first week of the illness, a fairly profuse macular rash appeared on the body. This persisted during the second week and gradually disappeared.

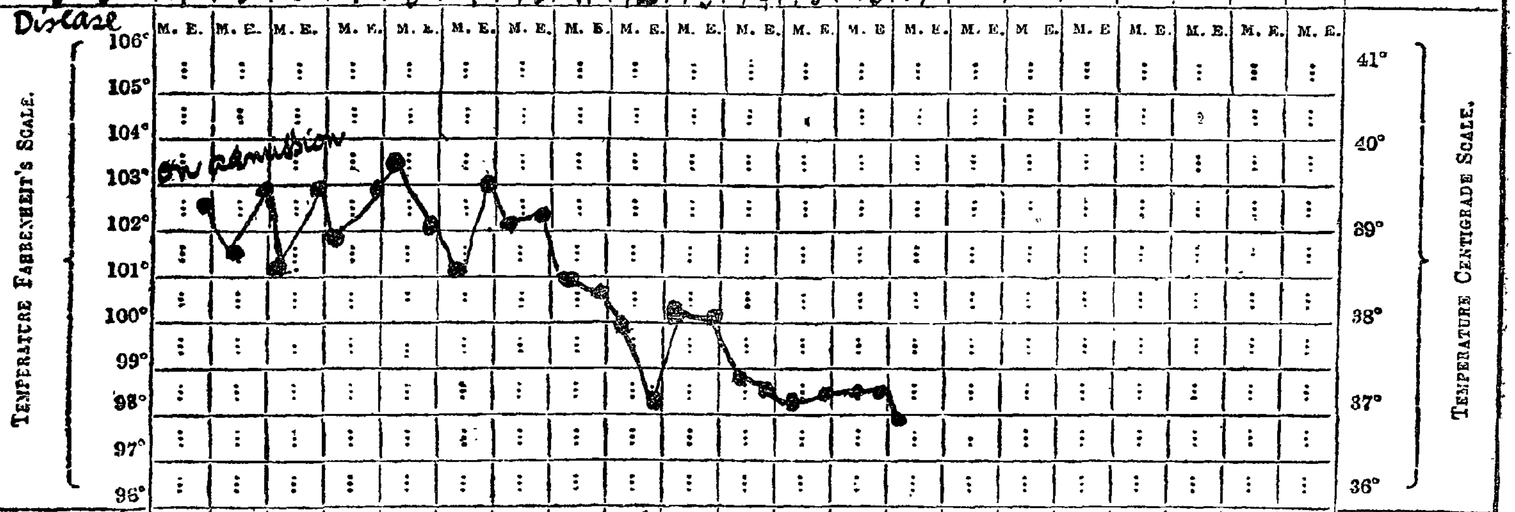
Throughout the illness patient was somnolent and drowsy and towards the end of the febrile period definitely stuporose, but the bowels remained regular and the abdomen soft and undistended throughout the whole attack.

The chart reproduced is typical of a mild case of tropical typhus.

It was not known that strains of *B. proteus X 19* were available in Madras and a blood sample taken at the height of the disease was

CLINICAL CHART OF TEMPERATURE, Etc.

Name Miss A.P. *Age* 22 *Disease* *Result*



Day of Disease	4	5	6	7	8	9	10	11	12	13	14	15	16	17								
	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	M. E.	
Pulse. M.		120	110	118	122	126	118	116	108	110	90	92	92	90								Pulse. M.
Pulse. E.	104	126	120	120	128	128	120	118	92	110	86	90	90	84								Pulse. E.
Resp. M.		28	26	30	28	30	28	28	28	26	24	24	26	24								Resp. M
Resp. E.	20	30	28	30	28	30	28	28	28	26	24	24	24	24								Resp. E.
Motions.																						Motions.
Urine, oz.																						Urine, oz.
" Sp. Gr.																						" Sp. Gr.
" Reaction.																						" Reaction.
" Chlorides																						" Chlorides
" Albumen.																						" Albumen
Day of Dis.																						Day of Dis.

haemolysed and septic when tested some weeks later. A further sample of serum obtained six weeks after the return of the temperature to normal agglutinated both *X 19* and the *K X* strains in 1 in 100 dilution (heated) but not 1 in 200.

It is now known that the typhus group of fevers is caused by the minute gram-negative *Rickettsia bodies* first discovered by Ricketts in Rocky mountain spotted fever in 1909. They have also been found in louse-borne epidemic typhus, in the endemic typhus of the United States (Brill's disease), in Tunis fever of North Africa, in tabardillo of Mexico, in the bush and urban types of tropical typhus in Malaya and in Japanese river fever.

The finding of Rickettsia bodies in the tissues of patients as well as in the various arthropod vectors concerned has given rise to the conception of a world-wide disease originally infecting rodents—which serve as a reservoir—and modified in virulence by local conditions and by passage through the arthropod vector concerned.

Upto the present time lice, fleas, ticks and mites have been incriminated as carriers.

Wilson of Belfast originally discovered that a bacillus of the proteus type is found in the urine in a certain proportion of cases of louse typhus and that the serum of typhus patients agglutinates it. His observations were extended and amplified by Weil and Felix working with the organism *B. proteus X 19* and the reaction now bears their names.

The relationship of *B. proteus* to the *Rickettsia bodies* is not yet fully understood, but the agglutination reaction of the serum of patients suffering from the different types of typhus fever towards *B. proteus X 19* and various other strains of *B. proteus* such as the Kingsbury strain (*B. proteus X K*) is proving of the greatest importance not only as a means of diagnosis but in immunological and epidemiological work.

Despite all the usual clinical and laboratory tests hitherto commonly employed, quite a fair proportion of our fever cases are not accurately diagnosed or classified.

Many of the shorter fevers of this nature fall into the DENGUE-SANDFLY group. For fevers of over seven days duration with completely negative laboratory findings we must now reckon with the possibility of infection with typhus-group fever, and serum taken in the second week should be sent to the King Institute or other well-equipped laboratory for

agglutination against several strains of *B. proteus.* Even with negative Weil-Felix findings or with a comparatively weak agglutination titre the disease may be diagnosed on clinical grounds if there is continued fever for 12 to 14 days with a sudden onset and a rather abrupt termination with negative tests for enteric group infection. The agglutination reaction is often weak or absent in the tick-borne variety, and much research yet remains to be done in this respect.

The diagnosis is most easily made on clinical grounds in fair-skinned people as the rash is easily seen. In a comparatively wide experience of louse typhus fever amongst Indian troops in Mesopotamia I found the rash very difficult to make out in some of the darker-skinned races of South India. In them the rash was most easily observed on the back (and on the limbs after the application of a rubber band or sphygmomanometer armlet for a few moments).

The rash of tropical typhus is as a rule less pronounced than in louse typhus and should be correspondingly more difficult to find in dark South Indians.

If readers will, therefore, consider the possibilty of TYPHUS GROUP infection in fevers lasting over seven days and ending about the end of the second week the diagnosis of a certain proportion of, at present, obscure cases will be cleared up.

I shall not enter into the details of symptomatology as these are fully dealt with in the numerous modern textbooks, notably in the 1935 edition of Rogers and Megaw's *Tropical Medicine.*

Fortunately the tropical non-epidemic varieties of typhus tend to be less severe than is the louse-borne epidemic variety, though the tick typhus of North India is more virulent than it was at first thought to be by Sir John Megaw who originally described it. In all cases of typhus infection, however, when toxaemia is severe hpdrotherapy both external and internal is the most powerful therapeutic measure at our command.

The use of rectal salines and of intravenous salines has helped many apparently hopeless cases of louse typhus to turn the corner, and similar measures should be employed in severe cases of endemic typhus. If headache is severe and if neck rigidity is present, lumbar puncture often gives great relief.

GASTRIC NEUROSES *

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It is common knowledge that the nervous system is intimately connected with all the various functions of the body, stimulating or inhibiting particular organs or set of organs as occasion demands. It is easy to understand, therefore, how when the nervous system is diseased or deranged, many of the ordinary functions of the body are correspondingly affected. The digestive system is no exception to this rule. The vomiting in cerebral tumour, the acute pains and other gastro-intestinal symptoms which occur in the crises of locomotor ataxy are examples.

When we speak of neuroses of the stomach, we exclude gastro-intestinal manifestions of organic neurological lesions as instanced above. As Goodhart has said "If I am going to write a book on indigestion, I should first devote myself to a volume on diseases of the Nervous System". Again he has said, "It is no great exaggeration to say that there are only two forms of indigestion; that produced by over eating and drinking, and that due to a failure of nervous power". Under the term *gastric neuroses*, we include only such disorders of digestion which arise from a lack of nervous power.

These disorders arise in different ways. They might arise from functional disturbances of the nerves supplying the stomach. Excessive stimulation of secretory nerves produces hyperacidity. Similarly, stimulation of motor fibres increases tonus and produces hurried peristalsis. In these conditions there are no gross changes in the nerves, but only modification of functional efficiency, both in time and intensity. In some cases, loss of tone of muscle of the stomach is brought about by undernutrition, leading to stasis and the consequent train of symptoms. This is particularly so in neurasthenia. In these cases, the stomach is the primary source of dyspeptic troubles—to cure which the measures should be directed to the stomach first. Secondly, in many cases of nervous dyspepsia, the symptoms resemble those of actual disease of the stomach; but the influence of a general

* A Lecture delivered under the auspices of the Madras University Extension Board on Feb. 25, 1935.

mental state of excitablility is clearly shown. Medical men are familiar with the dyspepsias that arise from states of anger, anxiety or hope. The most important factor in all these cases is the mental state of emotion connected with certain bodily conditions known as hypochondriacal anxiety. Fear of the evil effects of food produces the anxiety that it may bring on severe stomach troubles, and then to the gradually increasing state of mental agitation. Thirdly, many cases owe their origin to a psychic hyperaesthesia which translates a perfectly healthy sensation into one of actual pain in the stomach; for example, the ordinary movements of the stomach, consciously or otherwise, cause eructation and vomiting. Fourthly, anticipation has a share in the psychogenic origin of many nervous gastric symptoms. For instance, the anticipation, or fear, of pain leads to the actual feeling of pain. From the fear of nausea, nausea even increasing to vomiting comes on. And, in recent times, in certain countries, the dread of cancer produces in nervous patients all the symptoms of gastric cancer. It would thus be seen that in the great majority of cases of gastric neuroses there is no functional disturbance of the gastric nerves themselves, but a morbid excitation of the psychogenic centres. The effects of this disordered stimulation are specially manifested in the functional activity of the stomach. These gastric symptoms are merely part of a general neurasthenia—" neurasthenia gastrica ".

The subjects of this syndrome are patients in whom nervous intensity is greater than physical capacity. They may complain of neuralgia of the head and face, fits of exhaustion and palpitation. They are generally restless, lean and bloodless. They are dyspeptic. They complain loudly and bitterly of the stomach, and anxiously enquire about the various drugs and methods for curing their indigestion. If these unfortunate patients are to be benefited by treatment, the physician should realize early that their nervous system is at fault and that the indigestion is only a part of the nervous upset. But not all patients are of the type just described. It is not unusual to see anaemic young women who are lazy and slow in their movements complain of epigastric pain, anorexia and vomiting. They have polyuria, tenderness of the spine, and contracted fields of vision. These patients are often likely to suffer from gastric neuroses.

CAUSES OF NERVOUS DYSPEPSIA

Whatever the pathology of the condition, it is chiefly by their causation that neuroses of the stomach are known.

1. Dietetic indiscretion after strenuous exertion. Young or middle aged man, after an unusually full and strenuous day, has a heavy dinner, and goes to sleep well tired. He wakes up early in the morning with severe pain in the abdomen. He is restless and feels bilious in the morning and is indisposed for the following week, or, if he had vomited when the pain was on he escapes further trouble. If after this unpleasant experience, he is careful next time after similar exertion, to go to bed with a very light meal postponing his proper dinner to the following day, he escapes altogether. The stomach failed on the first occasion because the nervous energy was exhausted.

2. A second group of cases arises as follows. A keen man of business who takes no rest; he drives himself and those about him at full speed; every disappointment, every mishap worries him; he becomes nervous, fretful, sleepless, and ends in becoming a dyspeptic.

3. A very large group of cases of gastric neuroses arises as in sympathy with disturbances elsewhere; these could be conveniently grouped as reflex dyspepsias. They are often the result of disorders of the pelvic organs, bile-ducts or gall-bladder, and the appendix.

It would thus be seen that nervous dyspepsias arise in diverse ways with a variety of presenting symptoms. Gastric neurosis, therefore, is generally polysymptomatic and cannot be "typed." The symptoms have to be dealt with severally.

SYMPTOMATOLOGY

Pain in the abdomen and other sensory perversions form the chief manifestations of gastric neuroses.

The pain in the epigastrium and abdomen often simulates that of organic gastric lesions, chiefly peptic ulcers. But a careful examination and a history of the evolution of the symptoms will reveal many points of diagnostic usefulness. The pain in gastralgia does not usually penetrate to the interscapular region. Any pain and tenderness that may be felt in the back is lower down, about the 9th or 10th dorsal vertebra. The area of tenderness in the epigastrium is large, unlike the very limited area of tenderness and hyperaesthesia in ulcers. The attacks of pain come on independently of food and are not relieved by alkalies or food. Ordinary restrictions of diet which generally relieve the pain of other gastric disorders fail in gastralgias. Many patients with disorders of the stomach

complain of pain soon after taking food. But the degree of pain complained of by some neurotic young women is more unreasonable, to swallow a few spoonfuls of food being sometimes a severe torture. Such is never the case in patients with peptic ulcers.

Sensory perversions.—In neurotic patients, gastric distress does not always appear as pain. It is often revealed as perverted organic sensations. These are as hard to bear as the pain itself. These perversions generally occur as a sense of sinking or as excessive hunger. The sense of sinking which is referred to the abdomen is peculiarly distressing. The patient experiences a feeling of utter exhaustion, the voice becomes weak and low, and the face turns grey—almost an attack of fainting. But the patient does not faint. The sense of hunger is acute and depressing. It is a feeling of want rather than appetite. For, food soon produces satiety, though not much relief. This false hunger may be experienced any part of the day, mid-forenoon, late afternoon, or midnight. Saline purgatives very often bring on this hunger. Sudden vascular dilatation is said to be the cause of this peculiar sensation of hunger and emptiness. In the diagnosis of these sensory perversions, particularly the sense of sinking, the sudden postural variations in the pulse rate is noteworthy. When the patient is standing or sitting, the pulse rate goes quickly up to 110 or 120 per minute, and on lying down, in a very short while the pulse rapidly falls to about 70 per minute.

Bulimia.—Different from false hunger is the voracious appetite which occurs in some neurotics. The hunger and the ability to eat and digest may be enormous in some of the patients; it might be easily worse than in diabetics. Similarly, bulimia has been observed in affections of the vagus—a neuroma was found in one case; it is sometimes observed in Graves' disease.

Vomiting.—Uncontrollable and recurring vomiting after almost every meal is complained of by many. Two adult men were admitted into my wards with this complaint. But the vomiting ceased while under observation. I have seen this vomiting in two children of the same family. The children got over it after some months. It is remakable that these patients keep plump and well-nourished in spite of the repeated vomiting. Obviously, considerable portions of the meal are retained. This symptom occurs both in the idle rich as well as in the hardworking poor. Nausea is not very common in these cases.

Flatulence.—Windiness is often the most troublesome symptom of those who suffer from neurosis of the stomach. Flatulence and eructations come on in storms with such noise and gust that one often wonders whence these symptoms arise. The intolerable feeling of epigastric distension has no relation to the flatulence. This feeling may be so great as to oblige the sufferer to undress without any measurable enlargement of the waist. These patients almost regularly wake up in the small hours of the morning with an uncomfortable feeling of distension, belch up boisterously for some minutes, and then go to sleep again quite comfortably. According to Goodhart, this flatulence affects the stomach in men and the bowels in women. In some women, who are otherwise sensible, the neurosis takes the shape of "volcanic rumblings and noisy upheavals of air". This might sometimes be so serious as to necessitate their giving up their jobs.

Anorexia Nervosa.—We owe this name to Sir William Gull. There is gastric anaesthesia in which the natural sensation of hunger is blunted or even absent. The patients soon get emaciated. They are generally young women who fall victims to the craze of thinning. Though emaciated, with nothing beside the skin to cover their bones loosely, they continue capable of occupations, interests, and even of efforts, which are astonishing in such frail bodies. Though they may live literally on thimblefuls, they often find fault with some other members of their families for not eating enough! In these women the neglected appetite gradually begins to disappear and the body is tragically though painlessly pined.

Extra-gastric symptoms.—In women, the caecum and ascending colon are constricted and may be felt like a sausage or a coiled snake. This is due to spasm of the colon which often leads to troublesome constipation. I have known a case of a young woman of 32 who was suffering from the hyperacid syndrome. Eight years later, she developed severe attacks of pain, and a lump with tenderness was felt in the right iliac region. Appendicitis was suspected. On operation, appendix and caecum were normal. There were no adhesions.

In addition to these gastric and other abdominal symptoms, other nervous symptoms, such as increased psychic irritability, headache, vertigo and abnormal sensation in the extremities simulating neuritis, are often present.

DIAGNOSIS

It is generally not very difficult for an experienced physician to

mark out a case of gastric neurosis from the nature of the symptoms. One should take into consideration the general nervous state, the attacks of anxiety with marked mental flutter and the hypochondrial nature of the patients. The frequent variation in the symptoms, their dependence on the state of mind as agitation, diversion or recreation, and the co-existence of other nervous symptoms as headache, vertigo and palpitation often indicate the diagnosis. In all cases, a careful examination towards eliciting the possible presence of an organic lesion should not be omitted, especially since a careful examination is often the best treatment for neurosis. These unfortunates thereby develop a greater confidence in the ability of the physician.

Errors in diagnosis arise in a number of ways. (1) The symptoms may be of such an objective kind that one is compelled to assume the existence of organic lesions. (2) In neurotics, actual disease of the stomach may be masked by the general state of neurosis. (3) In some cases, though there may be no definite evidence of organic disease, certain abnormalities are still present, such as hyperacidity, hypersecretion, achlorhydria, and gastroptosis. These conditions should not be overlooked. But it is as easy to stress their importance too much, forgetful of the fact that very many people with these abnormalities go about in life without the least inconvenience. These are really excellent cases for treatment with suggestion.

Conditions which have to be eliminated before a diagnosis is made are: *carcinoma of the stomach*, *peptic ulcers*, *chronic cholecystitis* without jaundice. The points of difference between ulcers and neurosis have already been dealt with in the discussion of the symptoms. In all cases, a careful evaluation of all the symptoms, and watching the progress of the case and the result of treatment are evey helpful. X-ray examination of the stomach and the duodenum is essential. Cases which were considered neurosis, and put through various forms of treatment, have eventually been found on operation to present organic lesions, old ulcer cicatrices, and adhesions. That these lesions were really responsible for their trouble is proved by the fact that these cases improved and were sometimes cured by the operation. According to Clifford Allbutt, very trifling organic lesions like small umbilical hernias are likely to produce serious symptoms which could only be put down against neurosis if the small lesion be overlooked. In cases where organic and nervous symptoms are combined, diagnosis is only possible after thorough all-round examination and careful consideration.

PROGNOSIS

The prognosis depends on the degree of the underlying neuropathic state of the patient and his environment. Cases which are susceptible to suggestion generally do very well, especially if there is association of loss of flesh and undernutrition. These are the cases that do well on the lines of Weir Mitchell's treatment. But people in whom abnormal conceptions and general morbid psychic conditions are ingrained deep and long, and those in whom the harmful psychic influence continues to act, generally fare ill. If these influences could be removed, even apparently bad cases tend to recover. In all cases, there is an unfortunate tendency to relapse.

TREATMENT

The number of people who are victims of gastric neuroses is really very great. There is a tendency even now among medical men to ignore the complaints of these patients, once the diagnosis is made. The patient is sent out with a prescription or two of some one or other of the innumerable proprietary or patent preparations with an injunction not to mind his symptoms which are expected to clear up by themselves. It is true that we fail to find evidence of organic lesion. But there is no reason to forget that the distress of the patient is great and that the pain and other symptoms of which he complains is very real to him. The first and most important step in treatment, therefore, is a thorough and painstaking investigation of the case, including a complete history of the evolution of the symptoms. After this is done, if the patient is told that there is no structural lesion in his body, he is greatly assured, and often he does not trouble himself any further about his condition. On the other hand, over-anxiety and fussiness on the part of the doctor is very harmful to the patient. Whereas a definite course of treatment should be prescribed in every case, such exaggerations as the prescription of a rigorous diet with a string of dont's, or innumerable drugs, in the shape of drugs before and after meals with a "tonic" or two besides, are extremely harmful. Measures for general strengthening of the body and quietening of the nerves are often helpful. Change of environment, cold bathing, and friction to epigastrium are useful within limits. Electrical massages may be useful in obstinate constipation. Drugs have little use in these cases except as aids to suggestion therapy. He who pins his faith in drugs generally fails in these cases. It is the personality of the physician that achieves results.

HEADACHE: ITS DIFFERENTIATION AND SIGNIFICANCE*

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This paper, coming as it does from an ophthalmologist of more than ten years' standing, might be disappointing to you, since it will not deal specifically with ocular headaches as you might have expected, but will be concerned with the differentiation and significance of headaches in general. I hope to be excused for having made the content of the paper such for the following reason. I am now before you neither as an eye specialist who regards every headache as a symptom of eye-strain to be cured by means of spectacles, nor as a physician who for every headache would administer drugs without even so much as an enquiry regarding the visual condition; I have set myself the task of pointing out the many ways in which headaches may arise and the manner in each they may be differentiated.

Headache is the most common symptom complained of by ailing individuals; and it is most protean in its character, causation, and significance. Therefore, it confronts the physician as the most frequent factor in subjective diagnosis.

Nobody presumes that a diagnosis should ever be made on a headache alone, no matter how careful and refined a differentiation of its character might be attempted. This is true of every subjective symptom; and even of the sum total of all subjective symptoms in any given case. Symptoms are, after all, only the split half of the disease picture and they need to be supplemented by the other half (the objective signs) to complete the diagnostic data.

Ideally, no doubt, every patient, whether his chief complaint be a headache or any other subjective discomfort, should be subjected to a thorough routine examination which would embrace every possible source of mischief. But we know routine defeats case study. Case-taking does not consist in examining for symptoms that are not there, but in finding a

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clue and following it searchingly and intelligently. A good diagnostician "catches the scent like a hound and follows it, refusing to be drawn away by cross-trails; and diagnosing is like finding the way to a given house in a city of streets and we choose the most direct route from the point where we happen to be".

From this point of view, a symptom as common and as protean as headache must certainly be regarded as an important and valuable clue, or a sign-post, in diagnosis, whose character and occurrence suggest a line of investigation, and whose causation and significance merit systematic study. It is because of these considerations that some of the commoner causes and types of headache are here discussed.

I. NASAL AND SINUS HEADACHES

As nasal and para-nasal defects are probably the commonest affections among otherwise normal persons, so also are nasal and para-nasal headaches the commonest types of headaches. Particularly are the nose and sinuses to be suspected in those patients who complain of long-continued and troublesome headache without any apparent evidence of deep-seated internal disorder, such as alone could explain a persistent headache of a systemic nature.

(1) The simplest case of nasal headache is that which is due to pressure of the middle turbinal bone upon the nasal septum. Usually there is a deflected septum, and the middle turbinate, turgid and congested, becomes crowded against the corresponding part of the septum, causing a feeling of tightness in the nose and a headache, usually supra-orbital, prone to occur when the head is low or when the nasal mucosa is irritated by dust.

(2) Less simple, but almost as common, is the headache whose source lies not in the nasal passages, but in one of the sinuses which open into the nasal passages. The sensory innervation of the accessory sinuses is derived from various branches of the fifth nerve, and irritation by inflammation of the lining mucosa, frequently aggravated by pressure on the inflamed muco-periosteum by accumulating pus, causes severe and at times even agonising neuralgic pain.

The pain is often referred to regions by no means corresponding to the affected sinus. Thus, the headache of maxillary sinusitis is usually occipital; that of the ethmoidal is supra-obital or temporal, at the back of

the eyes, or is referred to the middle ear or mastoid; that of the sphenoidal sinus is usually vertical. A frontal sinusitis is usually associated with heaviness or pain over the sinus; but pain in this area is also frequently due to sphenoidal sinusitis.

These location factors, however, must not be taken with any too great particularization. The more one studies sinus disease, the more he realizes that they do not always hold good. While in many cases an uncomplicated frontal sinus disease will have a predilection for the supra-orbital region, an ethmoidal for the occipital, and so on, the reverse condition of affairs may be seen in many others. Moreover, inasmuch as more than one sinus is usually affected, the headache is not as a rule confined to one area.

A much more characteristic feature of nasal and sinus headache is the time and occasion of its occurrence. As previously stated, it is apt to occur when the head is low and when the nose is irritated by dust, etc. For this reason, patients usually complain that the headache is present when they wake up in the morning and diminishes or disappears after they have been up and about for a few hours (due, of course, to the accumulated congestion during the hours when they were lying in bed being relieved by the upright posture and the activities of the day), or, they state that the headache comes on during and after a motor or bus ride, when they are exposed to dust in the air. On the same basis, nasal and sinus headaches are usually intermittent. While they are present, under the conditions mentioned, they are often exceedingly severe; and during the intermissions, under favourable conditions, the patient is frequently quite free from the least sign of trouble. Finally, they can almost always be promptly relieved by the application of a little cocaine or adrenaline to the nose and turbinals, the temporary shrinking of the tissues causing alleviation of the pain.

It may be said in passing that in bad cases of sinusitis the irritation and pressure factors may be supplemented by a toxic factor, due to absorption of pus and toxins, so that the headache may partake of the nature of a toxic headache, presently to be described. And it is to be noted, further, that the amount of pus discharging from the sinus, or the absence of visible pus, is no criterion of the severity of the toxæmia; indeed, the opposite is frequently true, that, given an infection, the more pus is flowing the less the toxic effect.

II. OCULAR HEADACHES

Next in frequency to nasal headaches, come those which are due to the eyes; and these fall into two general classes, namely, (1) those that arise from functional eyestrain, and (2) those that have their source in some actual disease of the eye.

Of these, the former class is by far the most numerous. It may be said that something like 60 per cent of all people have some kind and degree of refractive or muscular error in the visual apparatus, and a goodly proportion of these uncorrected defects give rise to troublesome headaches. Here again, some generalizing about the localization of the pain is usually attempted in text-books. Hyperopia is commonly supposed to be associated with a frontal or brow headache; astigmatism with a temporal or occipital headache; and muscle imbalance with pain in the eyes themselves. But such localization does not always hold good.

As with the nasal and sinus cases, the most characteristic feature of functional ocular headaches is the time and occasion of their occurrence. Naturally, they depend upon the use of the eyes. They are worst after a heavy and continuous exercise of close vision, so that the patient usually complains that his headaches come on toward afternoon, last through the evening, and disappear after a night's rest. A correct diagnosis can often be made by the simple expedient of eliminating the cause by stopping completely the use of the eyes, and noting that the headache is relieved by this procedure.

Since headaches are more often due to the eyes than to anything else, the eyes should have careful study in every case of repeated headaches.

Since eye strain is very common in eyes with extra good sight (hyperopia), the presence of good vision is no ground whatever for thinking that a given case of headache is not ocular.

The location of the headache is not a very valuable guide. The severity of the headache does not depend upon the magnitude of the refractive error. It depends on the efforts, usually quite unconscious, made by the patient to correct it, and on his susceptibility, often hereditary. (I may add that this effort is usually greatest in small degrees of error, because where the error is high the patient commonly gives up all attempts to correct it.)

Nothing other than the harmless therapeutic test of wearing glasses is

conclusive, and even then one man's efforts may not be enough; often success follows only after several good ophthalmologists have failed to give relief; so elusive and difficult is the elimination of eyestrain in some cases, while it is so easy in others.

When we come to the second class of ocular headaches, namely, those due to ocular disease, it may be said that two eye diseases are responsible for the vast majority of this group—*iritis* and *glaucoma*. In both these, the headaches are usually one-sided, since only one eye is commonly affected; but this is not always true, especially in the case of iritis, which when due to systemic infection (rheumatic, syphilitic) may be bilateral.

(1) The headache of *iritis* is, as a rule, intense and deep-seated, although its intensity varies, with the acuteness of the disease. It radiates to the neighbouring parts, and is felt most severely in the supra-orbital region, i.e., in the brow. Perhaps its most peculiar characteristic is that it is worse at night, or in the dark; this is because the circular ciliary muscle, being relaxed in the dark, is pulled back like a string curtain and bunched on itself, giving rise to increased irritation and increased intra-ocular tension. The headache is usually, but not invariably, accompanied by redness of the eye, lachrymation, and photophobia; and an examination of the eye by means of focal illumination or the ophthalmoscope will disclose cloudiness of the media and a ragged, lustreless condition of the iris itself.

(2) The headache of *glaucoma* resembles very closely that of iritis, having, in fact, almost the same mechanism of production. Its location is identical with that of the headache of iritis, but its character is of a pressure type, seeming as though it would burst the eye and head, rather than of a deep-seated boring type. It varies all the way from the dull pressure ache to an agonising almost unbearable pain, often coming on suddenly at unaccountable times—in the middle of the night, or while the patient is watching a moving picture, for example—accompanied, as a rule, by great blurring of vision and flashes of coloured light. On the other hand, it may be mild and chronic, but almost always attended with perverted vision, such as blurring, yellow-green circles around lights, etc. Definite diagnosis can be established, of course, only by careful examination of the eye by one who is trained and experienced in such examination.

III. TOXIC HEADACHES

In a certain sense, all headaches are toxic, within the broad scope

of the term, for they all are the sensory expression of poisoning in some form or other of the tissues. Under this particular heading, however, we consider those headaches which are the direct result of the more or less general saturation of the body with toxins, either products of bacterial infection or of perverted metabolism.

Toxic headaches vary in degree from the slight, dull heaviness of a mild chronic infection to the intense stupefying ache of an intense acute infection as typified in typhoid fever or small-pox. But the difference is, after all, only one of degree and not of kind. For all toxic headaches, have essentially the same qualities and characteristics.

As would naturally be supposed, the toxic headache has no special localization. One of its cardinal characteristics is its generalization over the entire head; it may be light or severe, according to the degree of toxaemia, but it is usually steady and continuous, and deadly oppressive. Perhaps its most typical feature is the associated mental reaction. Of all headaches, it is the one which unfailingly and noticeably dulls the faculties and produces mental hebetude. Other headaches, by their severity, their location, or what not, distract and pre-occupy; the toxic headache alone blunts and dulls. By this one feature it can almost be diagnosed for what it is. Even the lighter forms, such as often accompany chronic tonsillitis, appendicitis, and other focal infections, exhibit this mental trait. And in the graver forms, such as we see in acute infections and uraemic nephritis, this often attains the degree of mental stupor.

IV. VASCULAR HEADACHES

The old conception of a congestive headache, commonly described as "a determination of blood to the head" (meaning the brain) is largely an exploded fallacy. It is quite likely that under certain conditions, such as hard and continued mental application, the blood is sent to the cerebral areas in large quantities; but, in view of the fact that the cerebral vessels are not furnished with vaso-motor nerves and of the mechanism provided in the brain for the equalization of pressure as between the arteries and veins, it is difficult to conceive of anything like congestion resulting from such a cause: certainly not enough to produce a headache.

It is highly doubtful whether even a general elevation of blood pressure is sufficient of itself to cause a headache—that is to say, by reason of mechanical intracranial pressure. Certain it is that thousands of people go through the later period of life with exceedingly high blood

pressures who do not experience any headaches therefrom. It is equally true that some cases of hypertension, both temporary and chronic, are attended with headache. Especially is this true of those cases of temporary hypertension, due to neurotic and endocrine disturbance, in which it may be that the rather abrupt rise of pressure, grafted on a neurotic temperament, does give rise to sensory phenomena in the head—which, after all, are neurological rather than vascular. But in the chronic conditions of hypertension which accompany nephritis, arterio-sclerosis and the like, the headache, when present, is more likely due to the toxemia of the disease itself than to the high pressure; and if it be vascular in character at all, it is far more likely to be a case of cerebral anæmia, due to interference with vascular supply, than a pressor condition. Toxic and neurological headaches are described elsewhere. The one type of vascular headache which can be considered a true vascular entity is the headache of cerebral anemia, whether it be the result of an impoverished or perverted condition of the blood itself, or of a mechanical interference with the distribution of the blood to the cerebral tissues. Such a headache is the classical "cry of a starving nerve for blood" and it has the classical characteristics of the pain which such a cry represents—similar is it in behaviour to the pain in other portions of the body where there is interference with the blood supply, for example, in the limbs when there is vascular claudication, or in the heart during an attack of angina pectoris.

The anaemic headache is intense and constrictive in character, often one-sided, but just as often vertical, giving a sensation of the scalp or skull being tightly gripped as in a vice or tied with a band, and attended by throbbing and beating. The muscles of the scalp are sensitive to touch and pressure, just as the thoracic muscles are in angina. The face is usually pallid, showing the blue veins plainly, and the skin is cool. The headache is more or less continuous, according to the underlying cause, but varies in intensity at different times, and is to some extent relieved by lying down.

It is highly probable that many of the headaches commonly diagnosed as migraine are in reality anaemic headache due to cerebral anoxaemia, although, to be sure, such an explanation does not help very much, since we are still faced with the problem of accounting for the anoxaemia in the absence of any apparent reason or mechanism for the condition. However

the important point is that the presence of a headache of this type furnishes a presumptive clue to investigation, and suggests the necessity of a blood-count and examination of the vascular system, and other lines of inquiry to explain the cerebral anaemia.

It may be said in conclusion, that the anaemic character of the headacbe can often be verified by an ophthalmoscopic examination of the eye ground, which frequently gives the typical anaemic appearances.

V. SYSTEMIC HEADACHES

Under this heading are classed those headaches, other than toxic and neurological, which come under the purview of the internist—headaches due to physiological and metabolic disturbances in the body functions, not the direct result of absorption of bacterial toxins.

(1) First, and perhaps the commonest, among this group are the headaches arising from causes in the stomach. They are chiefly the expression respectively of hyper-acidity and hypo-acidity of stomach contents, due probably to chemical irritation of the pneumogastric, the sensation being referred through the sympathetic to the fifth nerve. They are usually frontal in location, described by the patient as being "in the forehead", although they not infrequently extend to the temples, and occasionally even to the lower branches of the fifth, making the maxillary regions and even the jaws ache; as a rule, they are quite severe and even violent while they last; they are timed by the excessive acidity or otherwise of the gastric contents, and often disappear as abruptly as they come on.

But there is a characteristic difference in location between the headache of hyper-acidity and that of hypo-acidity. The pain of hyper-acidity headache was practically always centred at a point low on the forehead—about the root of the nose; that of hypo-acidity headache focussed itself at a higher frontal point—about the glabella. Both, however, were frontal and middle in location. Observation will, I think, corroborate the differentiation.

The gastric headaches are not, as a rule, difficult to recognise. Their almost fixed location, their temporary character, the relation that they bear to the function of digestion, and the co-existence of other signs of gastric disturbance, serve to establish the diagnosis. And, finally, the hyper-acidity headache is promptly relieved by the ingestion of a little bicarbonate of sodium, and the hypo-acidity headache

just as promptly by the administration of a little dilute hydrochloric acid. It should be remarked here, however, that although the relation between the headache and the acidity of the stomach contents may be simple enough, it by no means follows that the condition of the stomach is itself such a simple matter, in view of the modern understanding that stomach disturbances may not infrequently be the manifestation of pathology outside the stomach.

(2) It cannot be denied that intestinal stasis is frequently attended by headache. And this is true both of temporary and of chronic stasis. The mechanism of such headache is rather difficult to explain. The protagonists of intestinal toxæmia hold that it is a toxic phenomenon, due to absorption of toxic products from the bowel. In the case of temporary stasis at all events, this can hardly be so, because the headache quickly, almost immediately, disappears when the bowel is unloaded—altogether too prompt a result if we assume the headache to be due to a toxin-loaded system. It would seem more probable that it is a question of a neurological headache, in a patient of neurotic temperament. In chronic colonic stasis, the headache, when present, is the result of a combination of toxæmia and neurotic temperament, since these patients are usually both toxic and neurotic. Indeed, the colonic condition is generally the result, rather than the prime cause, of toxæmia and defective nerve energy; hence, the headache can well be attributed not so much to the colonic stasis as to the underlying condition which brought about the stasis.

At all events, the headaches met with in patients of this class are of the character described under toxic headaches and under neurotic headaches, and, while the intestinal stasis should be attended to as a "first aid" measure, such headaches call for careful investigation, and search should be made for an underlying toxic and neurotic factor that is causing both the headache and the colonic stasis.

(3) The *gouty* or rheumatoid headache is another of the systemic group. It is not really a headache at all, in the commonly accepted sense of the term, but a muscular and articular condition of the scalp and neck, similar to the myositis and arthritis which occur in other parts of the body, due to deposits. It has all the characteristics of an external muscular, or articular condition—not usually severe; mostly affecting the occiput and the back of the neck; painful on motion; still more so on motion after a long period of immobility; not attended by any of the mental hebetude

that accompanies a toxic headache; the scalp and neck tender and sore; and palpation revealing small nodules at the muscle insertions. Gentle massage of the muscles usually gives temporary relief.

(4) *Fatigue headaches* greatly resemble the gouty headache. The precise mechanism of muscle fatigue is still obscure. Whether or not we assume that fatigue is the expression of a toxæmia, it is certain that the headache of fatigue does not have the characteristics of the classical toxic headache, but rather those of the gouty myositis, with all the features just above described, except for the absence of nodules. And, it is a temporary affair, readily relieved by the simple expedient of rest. The headache here referred to is a purely physical condition, not to be confounded with the neurological headache commonly associated with mental staleness, "nervous breakdown", etc., which is quite a different matter.

VI. GYNÆCOLOGICAL HEADACHES

A lot of our former erroneous ideas about the headaches of women have, very properly, gone by the board. The old idea, that the physical life of a woman centres in and revolves about her pelvis, and that every ill her flesh is heir to is to be referred to her genital organs and functions, no longer prevails. We recognise nowadays that a woman is a complete human organism, just as a man is, and that most of her ailments, including headache are the same as those of a man. Even those headaches, which do have their seat of origin in her pelvis, or even in her distinctive female organs, are usually not different in their nature and mechanism from those of the male. They are chiefly toxic, vascular, and neuralgic.

Especially has the old-time conception of so-called reflex headaches (whatever that may mean) arising in some vague fashion from pelvic irritation been relegated to well-merited oblivion.

(1) However, granting that the majority of woman's headaches are toxic or vascular, still it cannot be gainsaid that there are certain types of toxic and vascular headaches to which a woman is peculiarly subject by reason of her feminine functions, and which are, therefore, properly classed and diagnosed as gynæcological headaches. Such a type is what may be called the menstrual headache from which a certain percentage of women habitually suffer at the menstrual period. It is probably a mixture of toxic, vascular, and endocrine headache.

(2) There are one or two types of headache which are peculiar

to *pregnancy*. One of these is the pituitary headache, due to enlargement and hyperactivity of the pituitary gland. It resembles the other intracranial headaches in behaviour, except for the eye symptoms, and particularly in its obstinacy and unamenability to treatment.

(3) More serious among these pregnancy headaches is that of pre-eclampsia, which, of course, is an expression of toxæmia, similar to the headache of nephritis, and exhibits the same characteristics. It disappears when the poison has been successfully eliminated.

(4) Finally, there is the toxic headache of puerperal fever, when bacterial toxins have entered the circulation. This is similar in all respects to the toxic headache of any other acute infection.

VII. NEUROLOGICAL HEADACHES

These naturally divide themselves into two classes: (1) those due to extra-cranial, and (2) those due to intra-cranial causes.

(1) *Extra-cranial Headaches* of a neurological nature are, necessarily, painful disturbances of the upper branch of the fifth nerve. They are either neuralgias or neuritises of this division and its sub-divisions. They are commonly susceptible of anatomical diagnosis, because they follow the distribution of the nerve, and are easily traceable to the areas supplied by it.

The neuralgias are usually due to focal infections or degenerative lesions in remoter structures supplied by branches of the fifth nerve, such as infected and decayed teeth, middle ear troubles, nasal and sinus affections, and tumours along the course of the nerve. They are characterised not only by their anatomical distribution but subjectively by their intermittency. They come and go, with no particular reference to time or occasion, except that temperature and other shocks frequently start them up. Moreover, they are often accompanied by tingling, numbness and other paræsthetic symptoms of nerve involvement.

The neuritises are ordinarily the result of some form of toxin, such as that of syphilis, rheumatism, or a focal infection. Their pain also follows the distribution of the nerve involved, but it is naturally more persistent and constant than that of the neuralgias. The area is tender and painful to palpation, and not infrequently the inflammation is marked by redness or slight eruption along the course of the affected nerve.

These extra-cranical headaches are not true headaches at all, but

facial and brow pains, and are perhaps the easiest of all such aches in which to recognise and demonstrate the superficial location.

(2) *Intra-cranial Headaches* are a totally different proposition. In this class are included some of the gravest, severest, and most intractable headaches known to medicine. They can be divided into two general classes, namely, *irritative* and *pressure* headaches.

Of the two, the *irritative headaches* are usually the more intense and agonizing. It may be said in general that they are always the expression of some form of meningitis, due to inflammatory irritation of the meningeal membranes. Like the peritoneum and the pleurae, under the influence of the infectious process which underlies the trouble, the meninges become easily irritated. The reaction is severe, often violent, producing a terrible headache which for agony and intolerableness is in a class by itself.

Pressure Headaches are the result of increased intra-cranial pressure, and are practically always due to the presence of some unwarranted mass in the brain, such as a tumor, an abscess, a gumma, a tubercle, or an organised blood-clot. The mechanism of the brain, as we have noted, is well provided against unequal pressure from purely circulatory disturbances, and as stated, it is questionable whether an *arteriosclerosis* or even a general *hypertension* can produce raised intra-cranial pressure. But this equalising mechanism itself depends upon the incompressibility of the intra-cranial fluid; and this same factor renders it impossible for even a small foreign mass to be present in the brain without at once creating an undue pressure within the cranial cavity. And the pressure on the sensitive meninges produces a severe and constant headache, not so agonizing as the irritative headache, because the pressure, while it wounds, also to some extent paralyzes the meningeal membrane, but bad enough.

The intra-cranial headache, whether irritative or pressure, has certain characteristics which should serve to distinguish it from any other type. Its severity and constancy alone should arouse suspicion of its nature: by which is meant the combination of constancy and severity. It hardly ever lets up, day or night. And, what is still more to the point, it yields to no alleviating measures, but only to deep anæsthesia or to opiates; often not even to the latter in quantities that are safe to be given.

Fortunately, for diagnosis, however, the identification of a cerebral headache does not rest upon the headache itself. It is always attended by certain eye symptoms which make its diagnosis certain. In the case

of the irritative brain headache, one or other, sometimes both, of the pupils is dilated; in the case of pressure headache, one or other, or both, is contracted. Usually, the dilation or contraction is unilateral, so that we have an unequal size of the two pupils. The ophthalmoscope will reveal further eye signs, an optic neuritis or a choked disc which respectively accompanies cerebral inflammation and cerebral compression.

Ordinarily, cerebral headache is attended by other symptoms such as vomiting (of the projective type) and inco-ordination.

The performance of spinal puncture is often followed by a headache, lasting for several hours, sometimes for a day or so. The reason for this is not quite clear, but it is highly probable that after the abstraction of cerebro-spinal fluid there is a rapid and temporary over-production of watery fluid in the cavity, similar to what takes place in the anterior chamber of the eye, causing a temporary hypertension, which becomes relieved as the fluid is reabsorbed.

Mention must be made of the neurotic headache, concerning the precise nature and mechanism of which, like that of all other neurotic phenomena, an explanation is difficult to find. And the character of such headaches is as vague and variable as that of all neurotic symptoms. The general rule of all neurotic syndromes may be applied equally to neurotic headaches, namely, that they have no definite character which can be diagnostically related to any definite pathology; and this is, perhaps, the chief factor in their identification. Only one headache of this class seems to have a constant character and location, and that is not, strictly speaking, a headache at all. I refer to the aching at the back of the head and neck which frequently accompanies a true case of neurasthenia.

VIII. SYPHILITIC HEADACHES

Although the headaches of syphilis do not differ in essential nature and mechanism from other headaches—being chiefly neurological headaches with their seat in the cranium—yet a special word about them may not be out of place, with a view to emphasizing their importance as a diagnostic clue to a syphilitic infection. As in all of its other manifestations, syphilis is protean in its production of headaches, giving rise in various cases to practically any and every type.

In its early stages, particularly the secondary stage, syphilis is frequently attended with a toxic headache (similar in character to that

in any mild infection) whose true significance is apt to be overlooked, especially in these days of exaggerated attention to focal infections. Later on, when the syphilitic process attacks the bones and meninges of the cranium, it produces irritative and osseous headaches, similar to those described under intra-cranial headaches.

In the *American Journal of Syphilis, April 1928*, the headaches of syphilis in its later developments are classified as follows:—

(a) *Meningeal type*: Headache is almost an invariable accompaniment of this type of syphilis, and often precedes all other symptoms.

(b) *Arterial type*: Headache may also be for a long time the only symptom of the arterial type of the disease.

(c) *Gumma*: Inflammation of the cerebral meninges is often attended by the formation of gummata and plastic exudates, the usual location of the gumma being at the base of the brain or over the front parietal region.

(d) *Cranial nerves*: Headache and neuralgia frequently attend syphilitic infection of the cranial nerves and the cervical plexus.

(e) *Persistent, periodical and nocturnal headaches* call for searching physical and neuropsychiatric examination for the possible detection of syphilis.

THE RELIEF OF HEADACHE

It is not my intention in this paper to suggest the treatment for headache. My purpose has been only to insist upon the role of headache in diagnosis, and it is in its diagnostic aspects that the subject is here presented and analysed. Nevertheless, in actual practice, headache, aside from its diagnostic value, often assumes the proportions of a distressing ailment *per se*, and the mission of the physician requires him to take measures for its relief.

A consideration of what has been said will show that, with the exception of those due to the eye and nasal structures, headaches, etiologically considered, fall into three general classes: toxic, vascular, and neurological. And whatever rational measures are undertaken for their relief must reckon with whichever of these three factors that lies at the root of the trouble.

10/8, BROADWAY, MADRAS.

GLEANINGS FROM MEDICAL LITERATURE

4 SPINAL ANAESTHESIA; NERVOUS SYSTEM SEQUELAE

GEORGE H. HYSLOP

Surgery, Gynaecology and Obstetrics, Dec. 1933. p. 799

Various sequelae affecting the central nervous system are known, but are individually rare. Though the technique of spinal anaesthesia including the precautions against shock is now standardised, statistics regarding accidents have chiefly dealt with fatalities from shock and do not take much account of sequelae affecting the central nervous system.

Spinal anaesthesia has been shown to have a haemolytic action on blood cells and a myelolytic action on the tissue of the cord. Though the mechanism of the fall in B. P. under special analgesia is not yet quite clear, it is reasonable to expect that an unduly great and rapid fall would predispose to cerebral vascular disturbances.

Spinal anaesthesia should not be used in individuals with poor vaso-motor tone. The possibility of some of the sequelae at least being due to infection would appear to be true.

The complications may be classified as:

I, *Local* effects, due to trauma caused by needle or the action of the drug on the nerve roots or cord tissue, are *cauda equina lesions, conus lesions, myelitis of the lower segments of the cord and spinal claudication.*

The cauda equina lesions are of the radicular type.

II. *Distant focal lesions.*

(*a*) Cranial nerve lesions. Of these extra-ocular muscular paralysis is most frequent, the external rectus being most often involved. The exposed position of the n. abducens would appear to account for this. Primary optic atrophy and nystagmus have been noted.

(*b*) Brain and brain-stem lesions. 2 cases of hemiplegia have been reported.

(*c*) Of the spinal cord lesions, diffuse ascending haemorrhagic myelitis, the Brown-Sequard syndrome and brachial nerve involvement have been mentioned.

III. *General.* Headache and stiff-neck for a few days are common and should not be regarded as either complication or as sequelae of spinal anaesthesia.

Infective meningitis is due to accidental infection during injection.

Encephalitis and aseptic meningo-myelitis have been reported,

Frequency: Headache as a transient phenomenon occurs in about 6% of cases; central nervous system sequelae in 0.5% of cases. Extraocular paralysis occurred in 5 out of 2000 cases (Reber).

Prognosis: The extraocular paralyses have a good prognosis. The toxic or aseptic meningo-encephalitic reactions seem to be benign. There is no evidence that any one of the various cocaine derivatives used predispose to central nervous system sequelae. Ocular paralyses are more apt to occur after stovaine than after novocaine probably due to the slow excretion of stovaine.

Treatment. The haemolytic and myelolytic action of the drugs used and the possibility that individual non-anaphylactic idiosyncrasy may predispose to sequelae, justify spinal drainage after operation, to remove any uneliminated and unfixed drug. Drainage has been suggested also to reduce the headache reaction.

M. K.

5 A LOWERED MORTALITY IN ACUTE APPENDICITIS

E. E. ARNHEIM AND H. NEWHOF.

Surgery, Gynæcology and Obstetrics, Aug. 1934.

These two writers discussing this subject are of the opinion that intraperitoneal suppuration and not some distant complication is the usual cause of death after operation for acute appendicitis. Death should occur only rarely after operations for acute phlegmonous appendicitis, appendicitis with local peritonitis, or appendicitis with abscess, unless diffuse peritonitis also exists at the time of operation. The factors contributing in varying degree to the lowered mortality are discussed under several headings.

Although all patients with a 24 hours or less history are operated upon promptly, on the second day and after, operation is deferred for some hours or longer depending on the condition of the patient. Dehydrated and shocked patients are placed on an intravenous drip of 5% glucose in normal saline solution. In cases of appendicular abscess, operation as a rule is postponed for several days, and is usually performed when the pulse rate and temperature are more or less normal and the mass well circumscribed.

On the question of anaesthesia the writers are of the opinion that spinal anaesthesia causes shock which lowers general resistance to infection and in that sense invites spread of infection. They recommend the use of avertin anaesthesia in the majority of cases.

A large incision which allows of full visualization is recommended. A rectus splitting incision in the majority of cases, or a McBurney's with severing of the muscles to provide better exposure if necessary, is generally employed. However, routine incisions which disregard the situation of the lesion are considered illogical, and whenever a mass is felt the incision is placed directly over it.

Tube drainage is considered harmful; it is said to invite spread and maintenance of infection, as also to cause post-operative foul discharge and sloughing. Packing by strips of idoform gauze of varying width is used in abscess cases as well as in non-abscess cases if appreciable purulent exudate about the appendix exists. Drainage is particularly stressed when the infected area is behind the caecum against the posterior parietal peritoneum. Rubber dam is used to separate gauze from intestines and the abdominal wall is closed in layers but not tightly about the drains. These drains are not disturbed until loosened by discharge. Their gradual shortening is usually begun about a week after operation. Infection of the abdominal wall is rare and discharge is rarely profuse and never foul.

Intravenous drip mentioned before is maintained after operation until the general condition is satisfactory. It is believed that the recovery of a number of patients is due to this procedure, Rest, if necessary with adequate amounts of opiates for that purpose, is enjoined. Cathartics are contra-indicated unless the patient is convalescent.

Finally, when the patient is not doing well after the operation, some infection related to the operative field must be searched for, and the timely drainage of a subphrenic abscess. an encapsulated intra-peritoneal collection of pus or a retroperitoneal phlegmon may prove to be the vital factor in the recovery of the patient.

P. G. R.

6 CHRONIC APPENDICITIS AND THE RIGHT LOWER QUADRANT SYNDROME

CHARLES W. LESTER.

The American Journal of Surgery, August 1934.

The author says that chronic appendicitis is a misnomer as true chronic inflamation is seldom found and the adjacent intestine is often as much at fault as the appendix in the production of symptoms. It should be remembered that normally the appendix, caecum, and terminal ileum are capable of a certain amount of movement, and that the lumen of the appendix is relatively uniform and without constrictions. The appendix, being a vestigeal organ, undergoes fibrosis, which increases with the age of the patient, at the expense of the lymphoid tissue. The production of gastric symptoms in the right lower quadrant (R. L. Q.) syndrome is due to the same sympathetic innervation. The true pathological changes in the appendix are not discernible to the pathologist, but to the surgeon they are visible in the form of abnormalities which interfere with the free exit of material from the lumen of the appendix and the normal mobility of the appendix, caecum and terminal ileum. Semisolid intestinal contents enter the appendix and pass the partial obstruction where they lose some of their moisture, become solid, and form faecal concretions. Kinks and adhesions leading to constrictions often lead to acute inflammation, whereas adhesions involving the caecum and terminal ileum have not been sufficiently recognised as the cause of chronic symptoms which act reflexly. These gross abnormalities seem to be more responsible for the symptoms than the histological ones. Mesenteric lymphadenitis sometimes simulates chronic appendicitis.

These pathological changes are responsible for three poorly defined clinical groups:

1. Cases with a definite history of one or more previous attacks of acute appendicitis.

2. Cases with right lower quadrant pain as a dominant symptom.

3. Cases complaining chiefly of gastric disturbances. variously described as heartburn, indigestion. sour stomach, etc.

Constipation and R. L. Q. tenderness are common to all groups.

In the first group, the results of operation are good. It is generally conceded that one attack of acute appendicitis predisposes to another unless the appendix has been removed, and the recurrent attack is of the fulminating type. The symptoms of the acute attack are fairly typical, and the chief source of failure is in the improper interpretation of the history.

In the second group, the pain may be dull and intermittent, or dull and relatively constant, but subject to exacerbations. There may also be digestive disturbances such as constipation, nausea and vomiting, hyperacidity, or general epigastric distress.

In the third group, epigastric pain often of the hunger-pain type is present in about half the cases. R. L. Q. pain is also present but is not a constant finding. There is R. L. Q. tenderness. The symptoms resemble those of post-pyloric ulcer. X.ray examination is of great value, for where the X-ray demonstrates both pylorospasm and R. L. Q. abnormality with or without post-pyloric ulcer, operation on the right lower quadrant alone—removal of the appendix and the release of adhesions—gives complete relief in a large percentage of cases. An appendix which remains filled for 3 days or more, or one that is angulated and fixed, is considered abnormal.

Constipation, ovarian disturbances in women, hypothyroidism, urinary disorders, gall-bladder disease, and neurotic conditions are frequently confused with R. L. Q. syndrome.

The treatment of chronic appendicitis is operation. The McBurney incision with its extensions or the lower right rectus incision (or any of its modifications) may be used. After exploring the abdomen, appendectomy is done. When membranes and bands exist they should be carefully divided and the gut freed. The after-treatment of these cases is also important and any symptoms should be adequately treated.

K. R. C. H.

7 CHRONIC AMOEBIASIS

"EDITORIAL"

Journal of the Indian Medical Association, April '34.

Amoebic infection is an important cause of a considerable degree of morbidity amongst the people. The usual manifestations of the infection are acute dysentery with passage of mucus, blood, and pus in the stools. But this acute phase sooner or later passes into the chronic stage in the absence of efficient treatment. Chopra reports that a routine examination of the stools of patients has shown an incidence of 12 per cent of amoebic infection. Cases are not infrequent where the infection has at no time shown any acute symptoms, being chronic or latent from the beginning. Such cases constitute the *carriers* who are the chief agents in the spread of the disease.

The only way to control amoebic infection in a community is to spot out the acute, the chronic, and the latent cases as early as possible and to institute the most efficient anti-amoebic treatment till a complete cure results. It is also essential that contamination of food and drink through contact with the excreta of carriers must be prevented; this is a public health problem involving adequate treatment of the sewage before its final disposal.

The early diagnosis of chronic amoebiasis will depend upon the physician's suspicion of its presence in cases of vague abdominal discomfort with or without definite pain, flatulent dyspepsia, diarrhœa, hepatic enlargement with tenderness, and on the thoroughness of his search for the vegetative or the cystic forms of the parasite in the stools. In some cases the presence of *E. histolytica* cysts in stools may be a mere coincidence and not the cause of the aforesaid symptoms. It should be emphasized that the manifestations of amœbiasis are protean; duodenal ulcer, cholecystitis, and pulmonary tuberculosis may be simulated. Abscesses of the liver and the lung are not infrequent complications; but they are very rarely seen in the brain, spleen and kidneys. The diagnosis of amoebiasis should be confirmed by the detection of

E. histolytica in stools, a specimen of which should preferably be obtained after the administration of a saline purgative and examined as soon as possible after evacuation at body temperature. In some cases clinical diagnosis may be made from a visualisation through the procto-sigmoidoscope of typical white mucus-capped small round ulcers on the mucous folds of the rectum and lower sigmoid with intervening areas of healthy mucous surface; it may be confirmed by an examination of a smear from these ulcers for the presence of *E. histolytica* cysts. In other cases an examination of the caecum and colon by opaque barium enemata may reveal characteristic patchy deformities in the region of the caecum, or irritability and hyperperistalsis of the terminal ileum and caecum. In a suspected case of chronic amoebiasis these modes of investigation are essential, otherwise many cases of early carcinoma of the bowel and chronic non-specific ulcerative colitis may escape detection. If in spite of negative bacteriological and radiological findings, a diagnosis of chronic amoebiasis be justified on clinical grounds, the appropriate treatment may be continued for at most a fortnight. Absence of definite clinical improvement warrants a revision of the diagnosis.

The treatment of amoebiasis is a complex problem. There is no doubt that some great advances have, in recent years, been made in the therapeusis of amoebiasis but more still remains to be done. There is almost universal agreement that *emetine* hydrochloride, strongly advocated by Rogers in 1912, is the drug of choice in acute amoebiasis. But accumulated experience has shown that only one-third to half the patients are cured by emetine, whereas the remainder pass into the chronic stage. The reasons are (1) emetine is a toxic drug which cannot be used in sufficient dosage for fear of myocardial injury and peripheral neuritis, (2) it fails to exert its action on the amoebae lying deep at the base of the ulcers, and (3) the acid reaction of the intestine interferes with the efficient action of emetine. Hence, other drugs were tried and *emetine bismuth iodide* introduced for oral administration; it is also a toxic compound which fails to eradicate the infection in a large number of cases in spite of two or more courses. Next, the organic *arsenical preparations* such as stovarsol, treparsol, and carbarsone were recommended for oral use. Clinical trials have definitely demonstrated a higher percentage of cure with arsenical preparations in combination with emetine than with the latter alone, but the danger of arsenic poisoning should be remembered; toxic erythema is the commonest, and sometimes exfoliative dermatitis and peripheral neuritis occur. Carbarsone has been reported to yield very promising results without any untoward effects. Chopra has recently studied the action of this compound in a series of 31 cases of chronic intestinal amoebiasis in doses of 3¾ gr. in gelatin capsules twice daily for a period of 10 days, and has reported 74·5 per cent cures. He points out that the proportion of probable cures to failures in this series was 5·75 : 1 as compared with 3·5½: 1 obtained with emetine bismuth iodide and 3·16: 1 obtained with kurchi bismuth iodide. Some *quinoline derivatives* such as Yatren (which contains 28 per cent of iodine) and vioform (which contains both chlorine and iodine) have lately come into prominence. Yatren in doses of four ¼ gram pills thrice daily for 15 days has been claimed to have given an apparent cure rate of 72 per cent. The only disadvantages are the cost of the drug and the diarrhoea that might ensue with such large doses. Vioform given in doses of ¾ gram daily for 10 days and repeated after an interval of a week is better than Yatren, and does not give rise to diarrhoea. Still more recently another drug, gavano, a derivative of ipecacuanha, is on the market, and Chopra says that it may be used orally without any toxic effect, but its amoebicidal property is much less than that of emetine bismuth iodide, kurchi alkaloids, and carbarsone, the cure rate being only 33·4 per cent; he,

however, points out that in cases of amoebiasis with hepatic complications, where carbarsone and kurchi alkaloids are completely ineffective, gavano may be tried. From an analytical study of the reports of various workers, the outstanding fact emerging is that carbarsone is probably the least toxic and the most effective drug for chronic intestinal amoebiasis without hepatic involvement.

K. R. C. H.

8 ALCOHOL AND AUTOMOBILE ACCIDENTS

Heise in the *Journal of the American Medical Association* describes a series of tests dealing with the effects of alcohol upon the driver of an automobile. Preliminary tests after administering 30 cc. of whisky showed diminished efficiency and accuracy in typewriting. The alcohol did not exceed 0.02 per cent in the tissues. A second series of experiments were then conducted, 150 cc. of whisky being given and driving tests conducted. These consisted of avoiding obstacles, backing, and substituting the hand-brake for the foot-brake in rapid stops. Disorientation was uniformly found, accompanied sometimes by exhilaration. The authors conclude.—

(1) Experiments indicated a measurable loss of efficiency and judgment, even when small amounts of alcohol are accumulated in the blood and urine.

(2) Considering a person sober as long as he can still walk and talk is responsible for the small value of present day statistics regarding the relationship of alcohol to automobile accidents.

(3) By analyzing consecutive accident cases involving injury and death, it is possible to throw light on the high incidence of week-end accidents and night accidents, and the surprising preponderance of accidents in which male drivers are concerned.

(4) In this series the drinking pedestrian was concerned with many accidents; in most cases a child or old person was struck by a sober driver.

(5) It is recommended that the chemical test for alcohol, which has been proved to be practical in confirming drunkenness and thus aiding in the conviction of drunken drivers, be adopted universally, at least to confirm the observations obtained by physical examinations.

P. G. R.

9 STUDIES ON RETICULOCYTES

P. Brahmachari (*Folia Haematologica* 52: 45, 1934) studied the reticulocytes in 50 Indian adults and 20 new-born babies within the first 12 hours after birth. His conclusions are:

1. The reticulocyte percentage in normal healthy Indian adults ranged between 0.17 per cent and 1.04 per cent, the average value being 0.5 per cent. In this series 48 per cent were above the average figure, 18 per cent were level with the average figure, and 34 per cent below the average figure.

2. The figures given by many writers about reticulocyte percentage in infants are much too high.

3. The highest reticulocyte percentage observed in new-born babies was 4.8 per cent one hour after birth.

4. In some cases there was an increase of reticulo-erythrocyte percentage 24 hours or more after birth as compared with that during the first 12 hours,

5. The lowest reticulocyte percentage during 24 hours after birth was 1·2 per cent.

6. In some cases, after a fall in the reticulocyte percentage there was a subsequent rise followed by a gradual fall.

K. R. C. H.

10 TREATMENT OF FRACTURE OF SPINAL COLUMN

F. JIMENO-VIDAL

Presse Medicale, 1933, xli, 752

The author describes Bohler's method of treatment for fractures of the vertebral column. The reduction is facilitated by local anaesthesia according to Schnek's method. With the patient in lateral decubitus. a lumbar puncture needle is inserted at a point 6 cm. from the apophysis of the injured vertebra (to the right if the patient lies on the right, to the left if he lies on the left) on a line perpendicular to the vertebral column, and introduced at an angle of 35 degrees towards the fractured vertebra, and 6 cc. of a 1 per cent solution of procaine hydrochloride is injected directly into the haematoma of the fracture focus. If the needle is bloody when withdrawn, it indicates that the haematoma was located, and an additional 5 cc. of the procaine solution is injected at the same spot; if it is not bloody one must try again to locate the haematoma.

For the reduction, the patient is placed in prone decubitus so that the groin is at the edge of the examining table. The upper part of the trunk is raised 25 or 35 cm. and the head and arms are placed on another table, so that the trunk is suspended between the two tables. A cushion is placed under the ankles, and the legs are strapped to the table in two places. In this position a hyper-extension of the spine is obtained. After a few minutes the upper part of the spine is raised still more by putting a box under the patient's arms. At the end of approximately 15 minutes the gibbosity has disappeared completely and the vertebral column is in hyperlordosis. Without the position of the patient being changed a plaster cast is immediately applied. It should extend anteriorly from the manubrium to the pubic symphysis and posteriorly from the 1st dorsal vertebra to the sacrococcygeal articulation, space being left on the sides to permit free movement of the arms and flexion of the legs to a right angle. The maintenance of the position of reduction depends on the support given to the manubrium, the pubic symphysis, and the point of maximum lordosis. The plaster cast must be retained until consolidation is complete and the vertebral column has recovered its capacity of support. This requires a maximum of 12 weeks in benign cases; in more severe cases 16 weeks or more are required. To prevent circulatory disturbances, muscular and osseous atrophy and articular rigidity, the patient is permitted to walk from the outset of the immobilization, and several times daily performs exercises consisting in extension of the arms and legs in all directions, flexion of the knees, flexion and extension of the trunk while lying prone on a bed with the pubic symphysis on the edge of a bed, and flexion of the legs in dorsal decubitus. An especially important exercise consists in carrying a sand bag on the head for 15 or 20 minutes, the weight of which is gradually increased from 5 to 40 Kg. When the plaster cast is removed the elasticity and supporting power of the vertebral column are as good as they were prior to the injury.

K. R. C. H.

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RESEARCH AND RECORD

Medical Education, like other branches of education, has been very much in the lime-light these last few years. It would be a mistake, however, to think that this zeal for change and reform is a recent one. But in all the changes that have been introduced from time to time, the only constant features noticeable are the increase of subjects for study and the ever lengthening period of instruction. And along with the addition of subjects and the lengthening period, examinations have increased apace in number and intensity. This is not peculiar to any one country. It seems to be almost universal. It almost looks as if the superiority of Medical Education in a country is to be measured by the number of years taken to complete the curriculum. India is no exception. Medical Education of the modern type is barely a century old here. At its inception it was a weak copy of what was obtaining in England. At the present day it is still fashioned on the English model, with a few modifications here and there. And the picture is almost a plausible reprint of the original. The educationist is satisfied that he is keeping abreast of developments elsewhere, but he hardly ever spends a thought on how the medical student reacts to this added load to his studies. It is argued by the teachers that researches in various branches of Medicine have been so intense and fruitful within the last quarter of a century that if the student is to have the advantage of these discoveries, it is necessary for the teacher to require more time and for the student to learn more subjects. But a little reflection would show the fallacy of this popular argument. It is true much work has been done, many searchings and researches; many facts and so-called facts have been recorded. But it is equally true that many statements have been found to be wrong and many results doubtful. Theories have been put forward and as hastily withdrawn. A teacher if he takes these few facts into consideration and ponders on it in his study, he would almost certainly find that the time needed to teach is not longer and the facts necessary to be learnt by the student are not more in number than what it was a generation ago. It

strikes us that the teachers demand more time so that they might think aloud in the class-room, thus making up for their neglect in their own study. Where experienced teachers are undecided, it would be wrong for us to be dogmatic. But that the students' troubles are increased and that the examinations are getting more nerve-racking, are admitted on all hands. It is, therefore, necessary before further changes are introduced into the curiculum of Medical Studies, to enquire if practitioners could not be trained equally efficiently with less subjects for study and fewer examinations to pass.

Another aspect of present day Medical Education requires consideration. It is freely admitted by all who have had anything to do with medical students in Madras that they are quite capable of getting through the stiffest examination just as well as students elsewhere; some would say that they do it sometimes easier than many others. Further, few would deny that the average graduate who goes out of our Medical Colleges is as competent to practise his profession as medical men elsewhere. But the difference at present between medical men of India and other countries is chiefly in the field of Research. Such of those young men who have been picked out for employment in institutes of research or in special duties requiring skill in laboratory technique or epidemiological surveys have done very well indeed. They generally do not suffer by comparison. But we refer here to the vast opportunities and the abundance of material in the larger hospitals which are allowed to run to waste. There are many young medical men employed in these hospitals. The routine work is done. But we do not hear much of observations made or results recorded which could be useful to others. This is not due to any intrinsic incapacity of the graduate, but to the training he gets while a student. Much of his time is spent in cramming unnecessary details. And in the wards, he spends most of the time in going round with the medical officers listening to what they are supposed to teach. This may be excusable in the undergraduates. But the best of them after qualifying, still spend their time in working for higher examinations. These examinations again are generally designed to test the capacity of the candidates for learning more details and more facts. If only these examinations could be more rationally ordered to test the capacity of candidates not merely to cram the contents of more books and journals but also to observe minutely and record systematically, we are sure the schools and colleges of Medicine would soon become centres of medical studies in the true sense.

The Madras Medical College would soon be celebrating its first centenary. Those who are organizing to commemorate the event in a fitting manner would confer a boon on the profession if they could utilize the occasion to sketch out a plan whereby research and record would receive greater attention.

PROBLEMS ON FILARIASIS *

In the course of three lectures, under the Travancore Curzon Memorial endowment of the Madras University, Dr. T. BHASKARA MENON has brought out the baffling problems in the understanding of FILARIASIS, a disease that is so important in South India. The lectures embody a review of most of the published work on the subject, besides including original work on the pathological aspects of filariasis.

In the first lecture, there is a short account of the history of the disease, which has been known in India, from the time of SUSHRUTA in the 6th century B. C. The puzzles regarding the peculiar endemicity of the disease are discussed in detail. The hyperendemicity in certain areas and the relative low endemicity in other areas, according to the author's views, cannot be explained by the accepted factors of high atmospheric temperature, high humidity and intensity of mosquito breeding. The question of transmission by infective larvæ entering the skin of the host from infected water is put forward as a mode of entrance, the larvæ escaping from the mosquito to the breeding grounds during oviposition or after death. The author lays stress on the genital type of the disease as being difficult to explain on the theory of mosquito transmission, since bites on the genitalia are far from common.

The variation in type met with in different localities is also brought forward, the upper arm type that is so common in Fiji being rare in India. Filariasis without symptoms is looked upon as a mild infection, a small dose of the infecting worm being insufficient to produce a blockage that is clinically apparent. Symptoms depend on the extent of filariation of the host.

The series of changes that occur between the passage of the larvæ from the proboscis of the mosquito and its actual presence in the larger lymphatics have yet to be studied. The difference between the genital types in the male and the female is regarded as due not to a descent from the abdominal glands but to differing depths of larval penetration.

* The Travancore Curzon Lectures 1934-'35.

The theories of microfilarial periodicity are discussed in the second lecture and Lane's hypothesis of cyclical parturition is reviewed in detail. The author's finding of three females in the testis, in the same state of development of the embryos, is brought forward as some evidence in favour of this view. The pathology of the lymphangitis is discussed. The author favours the view that the lymphangitis is an allergic or toxic reaction due to death of the worms or some toxic discharge from the worm. The bacterial theory of the lymphangitis is criticised.

The inflammatory changes round the worms are demonstrated in detail by microphotographs. The acute eosinophilic reaction round the worm is regarded as indicative of a toxic or allergic process and not due to secondary bacterial infections. The histology of the filarial granuloma and the later encapsulating reaction round the worm are brought out.

Histological studies show that endolymphangitis and fibrosing lymphadenitis result in lymphatic obstruction.

In the third lecture the pathology of elephantiasis is described. It is held that inflammatory changes are not necessary for the production of elephantoid oedema. On the basis of histological studies, elephantiasis is divided into an inflammatory and non-inflammatory type. Emphasis is laid on the dilatation of the superficial capillaries through which the collateral circulation is carried out.

With regard to diagnosis, besides the examination of the blood, the skin reactions are discussed, and the important advance of radiological diagnosis of filariasis is noticed in detail. The photographs and sections demonstrate how the calcifying worms can be located in filariated tissues. Mention is also made of a possible diagnostic importance of the adhesion phenomenon. The important advance in treatment that the lecturer emphasises is a direct attack on the worms after location. The local injection of sulpharsenophenamine into a tender spot, from which the lymphangitis starts, in early cases, is a method that has given promising results. A surgical removal of the worm is also discussed as another mode of attack in the early stages of the disease. Operative procedures in elephantiasis are also touched upon, and emphasis is laid on the new understanding of the pathology of the disease and the view of the importance of the parent worms in filariasis, in the problems of surgical treatment.